

total (non-lactational) energy expenditure comprises resting metabolic expenditure, energy expenditure induced by dietary intake, and active metabolic expenditure as a result of physical activity. It might therefore be suggested that the apparent discrepancy in energy balance between the two groups could be due to increased active metabolic expenditure on the part of the bottle feeders: whether this is the case I know not but the deficit of 2 to 3 MJ/day (the estimated energy requirement for lactation) could easily be accounted for by a moderate increase in physical activity of the bottle feeding group. Perhaps increased activity and a busy lifestyle accounted for the difficulty in recruitment of bottle feeders for the trial?

Furthermore, if it is claimed that lactation brings about changes in glucose and lipid metabolism, why did the authors not present information on respiratory quotients (or, more correctly, respiratory exchange ratios) in the resting and postprandial states? Such information must surely be easily obtainable from their indirect calorimetric data and might well help substantiate such a claim.

JOHN D CANNON

Department of Surgery,  
Ninewells Hospital and Medical School,  
Dundee DD1 9SY

**AUTHORS' REPLY**—Mr Cannon has raised an interesting point regarding differences in physical activity between our lactating and bottle feeding women. The only way in which one could attempt to answer this would be to measure total energy expenditure over one month using the new technique of stable isotopically labelled water ( $^2\text{H}_2^{18}\text{O}$ ) with precise measurements of breast milk output and energy. Nevertheless, if physical activity accounted for the energy differences we fail to see why the lactating mother needs then to reduce energy expenditure by other physiological adaptations as shown in our paper. We did not publish the respiratory quotients for this is an imprecise way of determining metabolism of a meal composed of carbohydrates, fat, and protein.

Finally, we would emphasise the central point of our paper that in any analysis of postpartum energy balance in lactating women the possibility of energy adaptive mechanisms must be considered.

R T JUNG  
P ILLINGWORTH  
P HOWIE

Department of Medicine,  
Ninewells Hospital and Medical School,  
Dundee DD1 9SY

### Hypomagnesaemic tetany associated with prolonged treatment with aminoglycosides

**SIR**,—Dr R Wilkinson and colleagues' lesson (22 March, p 818) is not new, although it apparently still needs learning. Increased urinary loss of magnesium because of slow excretion and accumulation of aminoglycoside in renal proximal tubular cells may be the sole explanation in some cases.<sup>1,2</sup> Secondary hyperaldosteronism may result, is also thought to cause hypomagnesaemia,<sup>3</sup> and is clearly an additional mechanism in others.<sup>4</sup>

Patients particularly at risk are those whose chronic underlying disease will probably necessitate prolonged or recurrent courses of treatment with aminoglycosides.<sup>4</sup> Hypomagnesaemia has been reported recently in a series of 12 young adults and teenagers with cystic fibrosis.<sup>5</sup>

Although aminoglycosides are usually said to be excreted rapidly by the kidneys, gentamicin has been recovered from the urine of a patient with

normal renal function 20 days after stopping treatment.<sup>6</sup> In another study renal accumulation was found to occur throughout treatment, with renal cortical concentrations at necropsy 100 times those in the serum and the kidneys accounting for 40% of total body gentamicin.<sup>7</sup>

Symptoms may develop during treatment,<sup>3</sup> but in many cases they do not occur until many weeks after treatment.<sup>1,4</sup> In patients receiving prolonged (for more than two weeks) or recurrent courses of aminoglycosides serum magnesium, calcium, and potassium concentrations should be monitored frequently both during and after treatment.

CHRISTOPHER J H KELNAR

Royal Hospital for Sick Children,  
Edinburgh EH9 1LF

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### UGC cuts and medical services: marginal effects

**SIR**,—I have read with interest the highly optimistic government response to the Social Services Committee's third report on the University Grants Committee's cuts and medical services (8 March, p 710).

Unfortunately the report is woefully out of date. Although the government response reflects the fact that at the end of 1984 the UGC cuts had produced only marginal effects on medical services—largely because clinical staff, together with the NHS, had protected such clinical services—the current situation is far worse than admitted previously. The past year has seen a continuing reduction in medical school funds, and further cuts (in some schools up to £500 000) are in prospect this year. With the NHS unable to fill the gaps left by the inevitable reduction in clinical academic staff the combined effect of the university medical school funding difficulties and the restriction on NHS funds will mean a major reduction in teaching hospital clinical services. Some London medical schools have already questioned whether they will have enough staff to teach and sufficient patients for students to see to fulfil educational requirements. It is facile of the government to pretend that all is well when such major changes have occurred and continue to occur. We must do our utmost to rectify this apparent government ignorance of the dire circumstances of our medical schools and teaching hospitals.

COLIN L SMITH

Chairman, Medical Academic Staff Committee

BMA,  
London WC1H 9JP

### On the state of health in inner London

**SIR**,—May I comment on Dr R Balarajan's "special pleading" for London (29 March, p 911)? All major cities have their inner areas, but in London the inner area is large enough to be easily identified for statistical analysis.

In Leicester I have made similar comparisons between postcode sectors (approximately 10 000 people). The same differences in mortality and morbidity can be shown—for example, perinatal deaths 205, deaths from accidents 274, premature deaths 163, off work because of sickness 231. All measures of mortality and morbidity are highly correlated with measures of social and economic deprivation.

The planners' responsibility is not the diversion of funds from one region to another, but it should be the identification of areas with special needs within districts and making sure that these areas receive positive discrimination in the allocation of health services.

J J JONES

Department of Community Medicine,  
Leicester Health Authority,  
Leicester LE1 6TP

### Analysis of authorship

**SIR**,—Dr Peter Morgan's leading article (8 March, p 646) about the Rock Carling lecture<sup>1</sup> has prompted me to write about an aspect of journal publishing that has interested me for some time.

I am interested to know whether any studies have been carried out on the analysis of authorship, as opposed to scoring on a rating system for single authorship, dual authorship, multiauthorship, etc.<sup>2,4</sup> If you could ever persuade authors to agree to it, it would be an interesting exercise to indicate, directly under their names on the paper, what part each played in the work. For example, (a) the original thought/idea, (b) the science, (c) the technical help, (d) being head of the organisation, (e) being head of the section/group/team, (f) providing samples/patients, (g) carrying out statistical analysis, (h) providing DNA probes, (i) providing photographic/art work, and, last but not least, (j) who actually wrote the paper. These examples are in no particular order, and I am sure there are others that could be added to the list.

Within a unit such as the one from which I write most people know who has contributed what in the production of papers, but that information is not formalised or readily available. Indeed, readers may know—again by personal knowledge and contacts—the analysis of authorship of papers outwith their own institution. Another aspect of this is the order in which authors appear on a multiauthor paper. Convention may differ from one group to another—for example, where a head of department will always put his or her name first or last; occasionally the names may be alphabetically listed. The *Lancet* puts authors' qualifications on the front cover of the issue, but in the paper itself these are not included. The *BMJ* does the reverse: the authors appear without their qualifications on the front cover, but these are included in the paper itself.

S M MOULD

MRC Clinical and Population Cytogenetics Unit,  
Western General Hospital,  
Edinburgh EH4 2XU

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### Correction

#### Profound hypophosphataemia in patients collapsing after a "fun run"

The letter by Professor D R Wilkie (8 March, p 692) referred to the paper by Dr G Dale and others published on 15 February, p 447. We regret that this reference did not occur in Professor Wilkie's letter.